Table 1 Lung epithelium integrity and respiratory health of children having followed a swimming baby programme

	Swimming baby (n = 43)	Other children (n = 298)	p value
Age (mean, SD), years*	11.5 (0.6)	11.5 (0.6)	0.98
Boys, n (%)†	22 (51.1)	150 (50.3)	0.92
Mother and/or father with asthma, n (%)†	6 (14.0)	58 (19.5)	0.39
Aeroallergen specific serum IgE, n (%)†	13 (30.2)	95 (31.9)	0.83
Total serum IgE (median, IQR), kUI/l‡	54.7 (24.6–162)	55.8 (21.9–175)	0.96
Serum CC16 (mean, SD), µg/l*	8.0 (3.3)	10.4 (4.2)	0.01
Serum SP-D (mean, SD), µg/l*	113 (42)	100 (45)	0.08
Serum CC16/SP-D ratio (median, IQR)‡	0.07 (0.05–0.12)	0.10 (0.07–0.16)	0.003
Asthma†	10 (23.3)	33 (11.1)	0.025
Recurrent bronchitis†	26 (60.5)	110 (36.9)	0.006

CC16, Clara cell protein; SP-D, surfactant associated protein D; IQR, interquartile range.
*Two sided unpaired *t* test; †χ^2 test; ‡two sided Mann-Whitney U test.

positive exercise induced bronchoconstriction test (based on a 15% fall of FEV_1).[3] Backyard multiple and logistic regression analyses were used to further assess associations between baby swimming practice and the outcomes by testing a total of 23 potential predictors, including classical risk factors (for example, gender, serum IgE, and family history of allergic diseases).

There were no statistically significant differences between the swimming baby group and the other children regarding the proportion of children whose mother or father had asthma, the mean levels of total serum IgE, and the prevalence of aeroallergen specific serum IgE (table 1). Children who had been swimming as babies showed a significant decrease of serum CC16 and an even more significant decrease of the serum CC16/SP-D ratio, adjusting serum CC16 for the permeability of the alveolar-capillary barrier.[5] In multivariate analyses, baby swimming emerged as the only statistically significant predictor of serum CC16 (partial $r = -0.14$, $p = 0.01$); this practice was the strongest determinant of the CC16/SP-D ratio (log transformed values, partial $r = -0.15$, $p = 0.006$). These effects were associated with higher risks of asthma and recurrent bronchitis, as confirmed by logistic regression analyses (adjusted odds ratio for asthma 3.0, $p = 0.01$; adjusted odds ratio for recurrent bronchitis 2.6, $p = 0.006$).

Our data suggest that swimming baby practice in chlorinated indoor pools can be associated with distal airways alterations predisposing children to the development of asthma and recurrent bronchitis. While an increase of serum SP-D reflects an increased permeability of the alveolar-capillary barrier, the reduction of serum CC16 means a loss of the Clara cells lining the terminal airways.[5] These effects might result from repeated inhalation of chlorination products, in particular of trichloramine, the irritant gas formed when chlorine reacts with organic matter brought by swimmers and that gives indoor swimming pools their typical chlorine smell.[2 3] A link between swimming as a baby and more frequent recurrent respiratory diseases has also been observed in a recent study.[6] Although these findings need to be confirmed by prospective studies, we recommend caution before regularly taking babies to poorly ventilated indoor pools where there is a strong chlorine smell.

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MMR Catch up Campaign: reasons for refusal to consent

Following the adverse publicity regarding MMR vaccine,[1] MMR vaccination rates have declined. Kingston had the highest uptake of MMR in London (83–87% in 2003–04), but is still below the national target.[2] A targeted MMR Capital Catch up Campaign was introduced by the London NHS for the estimated 90 000 primary school children (aged 4–11 years) susceptible to measles (received less than two doses).

We conducted a descriptive study looking at parents' reasons for refusing MMR vaccination, analysing retrospectively all returned consent forms, from 50 primary schools from the Royal Borough of Kingston (fig 1), between December 2004 and April 2005. Parents were asked to indicate one of three options: consent to vaccination; refusal of vaccination; or no need for vaccination (as child had already received two doses of MMR). They were also given access to evidence based information on the MMR vaccination. Parents who refused consent were invited to state the reason(s) why. For the purpose of the study, the forms were anonymised to ensure confidentiality. Consequently individual follow up of cases was not possible.

All children were targeted because of poor baseline data on previous MMR immunisations. We summarised the responses into 13 different categories (fig 2). Of the main reasons given, 23% stated they wished to be present with their children during the vaccination and would prefer to have the vaccination done at the GP surgery. The autism/bowel disorder controversy scored highly (16%), as did concern regarding side effects of the vaccine or previous reactions. Three per cent stated a medical contraindication, such as receiving immunosuppressive therapy or

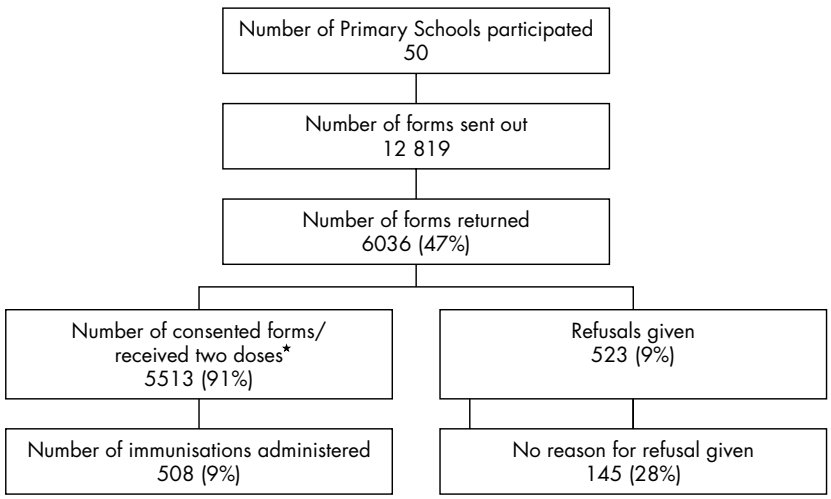

Figure 1 Profile of MMR Catch up Campaign in the Royal Borough of Kingston. There were no accurate baseline data on previous MMR immunisations; therefore all children were targeted in the 50 participating schools. Consent forms were given to children in schools to hand to their parents. Completed forms were handed back to school. For the purpose of the study, the forms were anonymised to ensure confidentiality. Reasons for refusal were analysed using Excel. *These two groups were counted together.

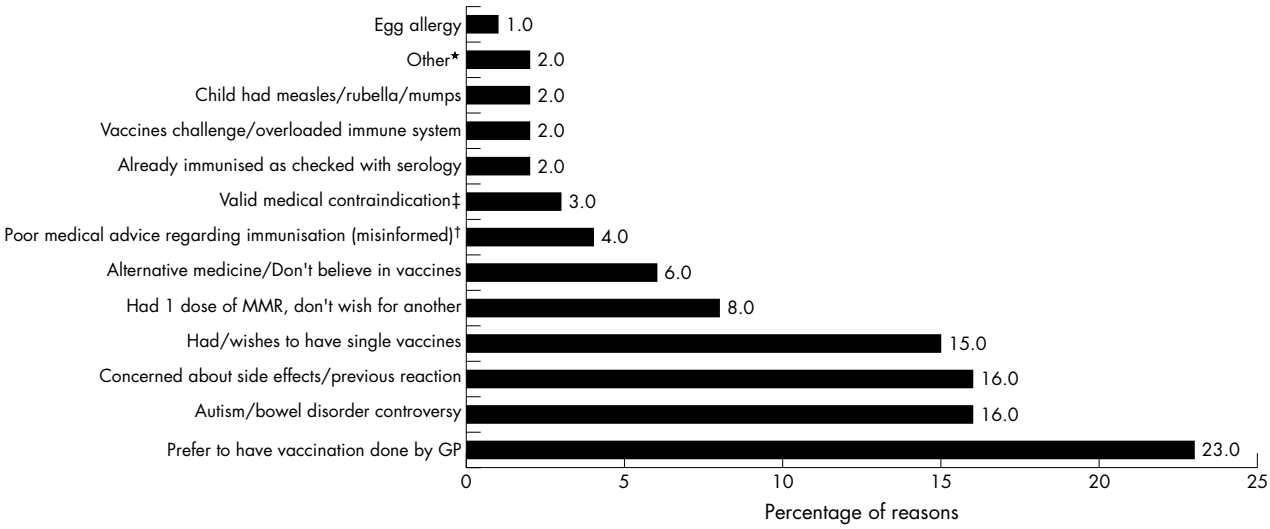

Figure 2 Reasons for refusal of consent to vaccination. *Objection to the munufacturing process of the rubella component, requests for serology prior to vaccination. †Egg allergy, multiple food allergies, family/personal history of convulsions. ‡Immunosuppressive therapy (chemotherapy, high dose steroid therapy, immunosuppressive drugs), immunoglobulin injections.

immunoglobulin injections, and 4% of refusals were from poor medical advice (fig 2). Only 9% of those who returned their forms (47%) did not provide consent for immunisation. We are unaware of the motives of parents who did not return their forms (53%). Of the target population, 2.9% gave reasons for refusal to consent.

The results from our study reflect the findings of similar previous studies,[3–5] namely "alternative views" on immunisation, the influence of the media, and mistrust of the advice of health professionals and the government. We suggest that intense educational programmes are set up locally for health professionals and the public, prior to any future catch up campaigns. Uptake could also be improved by arranging a more reliable distribution of the consent form, notice of intended campaigns, and a choice of venue for immunisation.

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This could be due to maternal testosterone…

It is my hypothesis that the "secular trend", the increase in size and earlier puberty of children, is caused by an increase in the percentage of women of higher testosterone within the population with time. Maternal testosterone has been connected with autism. I suggest the increase in autism, and other increasing disorders, that are occurring is due to the secular trend; I suggest increasing exposure of fetuses to increasing maternal testosterone is increasing autism (http://www.anthropogeny.com/increase in autism.htm).

This increase in maternal testosterone occurs in different groups of women of differing levels of testosterone. Therefore, the consequences of this fetal exposure will manifest as increases in different ways. For example, I suggest obesity, diabetes, and breast cancer are increasing because of this exposure in different women.

Dr Baron-Cohen[1] has demonstrated "mothers of autistic children often show patterns of brain activity more associated with men" (BBC comments on the internet). I suggest the connection of these types of mothers is that they may produce increased levels of testosterone.

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Author's reply

I welcome James Howard's letter prompted by my article on autism in your journal. The hypothesis that one risk factor for autism may be maternal levels of testosterone has several pieces of supportive evidence.

First, second-to-fourth digit (2D:4D) ratios[1] show sexual dimorphism related to testosterone levels, and mothers of children with autism have masculinised 2D:4D ratios. Second, mothers of children with autism have a hypermasculinised pattern of brain activity (measured by blood flow during fMRI while performing tasks which demonstrate sexual dimorphism in the typical brain).[2] Whether this reflects testosterone levels is not known, but there is a large literature from animal research demonstrating fetal testosterone levels shape sexual dimorphism in the brain. Finally, and most directly relevant to the maternal testosterone hypothesis, we have just completed a study surveying mothers of children with autism for testosterone related medical conditions. These mothers show increased rates of these.[3]

It is of interest that fetal testosterone levels (measured following amniocentesis) correlate inversely with social behaviour and directly with narrowed attention postnatally.[4] If it turns out that a fetus who later develops autism has high testosterone levels, and their mother does too, this could reflect shared susceptibility genes, or maternal testosterone acting as an environmental risk factor by crossing the placenta, or both. It is important to highlight that at present we only have a set of clues for both the fetal testosterone and maternal testosterone hypotheses in relation to autism, and that considerably more research is needed in this area.

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